

Advanced Dietary Fibre Technology

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13 Dietary Fibre, Carbohydrate Metabolism and Chronic Disease

David J.A. Jenkins, Alexandra L. Jenkins, Cyril W.C. Kendall, Livia Augustine and Vladimir Vuksan

13.1 Introduction

Historically, although this field owes much to Surgeon Captain Cleave and others before him, it was Denis Burkitt and Hugh Trowell who brought the idea of fibre and health together and into the public eye. Burkitt's main interest was in the connection between fibre and colonic disease (Burkitt & Spiller 1993), while Trowell (1993) carried the torch for the metabolic effects of fibre on carbohydrate and lipid metabolism and their related disorders. Many of the concepts they proposed found support in the work of Walker in South Africa, on whose findings they drew extensively.

As the debate progressed in the 1970s, many of the health benefits originally observed for a high vegetable fibre diet in Uganda became transferred to wheat bran for the Western world, possibly because of the visible immediacy of the colonic effect. Over the subsequent years, interest has shifted to viscous fibres, resistant starch and fructo-oligosaccharides – all of which fulfil the definition of fibre, as carbohydrates which enter the colon, but which have been excluded from the definition of fibre by those who consider that only cell-wall materials can be classed as dietary fibre. Nevertheless, they share many of the attributes of cell-wall polysaccharides, both in the small intestine and in the colon.

13.2 Effect of viscous and non-viscous fibre on glucose, insulin and gut hormones

Early on, it was shown that insoluble fibre sources such as wheat bran had little effect on glucose tolerance and the postprandial insulin response. However, the effects of viscous fibres on glucose tolerance and insulin responses was marked, and dependent on their viscosity (Jenkins *et al.* 1978). Studies using xylose excretion as a marker of the rate of absorption confirmed the slower rate of absorption with the more viscous fibres. These studies have been confirmed with other fibre sources, notably oat β -glucan (Metz *et al.* 1991). The mechanism appears to relate to impedance of solute movement through the unstirred water layer (Blackburn *et al.* 1984) and impedance to bulk phase diffusion of the products of digestion from lumen to the enterocyte (Flourie *et al.* 1984). Studies have also shown that the benefits of slowing the rate of absorption can be mimicked by sipping glucose over 3 hours rather than taking it in a bolus in 5 minutes (Jenkins *et al.* 1990). The concept of the advantage of spreading the nutrient load over time (e.g. nibbling versus gorging) has been shown to be of benefit in diabetes. Diabetics provided with small, frequent meals showed lower mean glucose and insulin levels over the day, than when the same amount of food was taken in larger discrete meals (Jenkins *et al.* 1992; Bertelsen *et al.* 1993).

Studies after gastric surgery have suggested that the so-called 'dumping syndrome' can be improved metabolically by viscous fibres which blunt not only the glucose rise but also the endocrine response, including insulin and the incretin or insulinotrophic gut hormone, gastric inhibitory polypeptide (GIP) (Jenkins *et al.* 1980; Leeds *et al.* 1981).

13.2.1 Effect of other fibre-like carbohydrates

True chemically resistant starches, RS₂ and RS₃, have no effect on the glycaemic response of the available starch (Jenkins *et al.* 1982a) when the resistant starch is accounted for by subtraction from the total starch in determining the test dose, i.e. the remaining starch gives a predictable high blood glucose rise. These starches are chemically resistant in that they require solubilisation with sodium hydroxide, etc. prior to analysis, and owe their resistance to hydrogen bonding. Entrapped starch (RS₁), which may be trapped by fibre reacts as slow-release carbohydrate (see below). This starch simply requires to be freed from its retaining physical matrix to be made 'available', e.g. by fine grinding.

Fructo-oligosaccharides have no major effects on the glycaemic response of accompanying carbohydrate, and have no glycaemic effect *per se*.

13.3 Fibre in whole food: food form and glycaemic index

Fibre may be one of the factors reducing the availability, or limiting enzymatic access to the starch. A portion of the starch may be digested so slowly that some enters the colon and is called resistant starch. It is not truly resistant starch, but is slowly absorbed, and thus unlike chemically resistant starches (RS₂ and RS₃) it results in a flattened blood glucose profile. Foods containing these fibre and starch matrixes are low glycaemic index foods and are more slowly digested *in vitro* (Jenkins *et al.* 1982a; Englyst *et al.* 1999). They have also been described as Slowly Available Glucose (SAG) sources (Englyst *et al.* 1999). Associated with prolonged free fatty acids (FFA) suppression, these foods also appear to improve the second meal glucose tolerance (Axelsen 1999; Jenkins *et al.* 1982b; Wolever *et al.* 1988). Examples of these foods are dried legumes, peas, beans and lentils, pumpnickel rye bread and bulgur or cracked wheat. In these cases fibre and food form may reduce the rate at which these foods are digested.

13.3.1 Fibre and glycaemic index: diabetes, cardiovascular disease and cancer

Fibre and its influence on glycaemic index, as a determinant of food form, may influence a number of chronic diseases. Viscous fibres may reduce serum lipid levels, secondary to inducing increased bile acid loss (Anderson *et al.* 1984; Lia *et al.* 1995; Jenkins *et al.* 1997). They also blunt the glucose and insulin responses. There are further means by which low-glycaemic index and viscous fibre-rich foods may confer benefit. Postprandially, they may reduce the level of oxidative stress and the production of free radicals generated after consuming refined carbohydrate meals (Geriello *et al.* 1998). Low serum cholesterol, postprandial glucose, insulin, insulin-like growth factors and reactive oxygen species may all therefore influence the risk for cardiovascular disease, diabetes and cancer.

Diabetes

Ironically, it is the insoluble cereal fibre which appears to offer protection from diabetes in the Nurses Health Study, the Health Professionals Study and the Iowa Women's Health Study (Salmeron *et al.* 1997a, b; Jacobs *et al.* 1998). In addition, low-glycaemic index diets or diets with a low glycaemic load (dietary glycaemic index \times dietary carbohydrate) were also negatively related to the development of Type 2 diabetes over a six-year period (Salmeron *et al.* 1997a, b). The glycaemic effect is understandable, but it is not as easy to see why the cereal fibre has an effect in reducing the incidence of Type 2 diabetes, unless it acts through associated nutrients such as magnesium or antioxidants. The effects of wheat bran on glycaemia are not marked. It is however possible that bran phenolics may be useful antioxidants of importance in diabetes prevention, or that other systems related to diabetes are favourably improved by cereal fibre.

In terms of disease treatment, low-glycaemic index diets have been shown to reduce glycosylated proteins in the majority of Type 1 or 2 diabetic subjects studied in over 13 trials of low-glycaemic index foods. One of the most recent studies also demonstrated a reduction in plasminogen activator inhibitor (PAI; Jarvi *et al.* 1999). This reduction of a haemostatic risk factor associated with thrombosis may have a definite advantage in reducing some of the risks of the complications of diabetes.

The effect of viscous fibres showed early promise in the treatment of diabetes; however, the lack of readily available palatable formulations has made further work in this area difficult. In addition, although some early insoluble fibre studies looked hopeful in terms of Type 2 diabetes treatment (Bosello *et al.* 1980), there have been no recent reports on the successful use of wheat bran in diabetes. There is also the question of whether fibre has a role in weight loss. In this area, recent data in obese boys indicate that favourable metabolic change and increased satiety accompany meals of reduced glycaemic load (Ludwig *et al.* 1999a, b).

Cardiovascular disease

Epidemiologically, wheat bran in a wide range of studies appears to be protective against the development of cardiovascular disease (Jacobs *et al.* 1998; Lui *et al.* 1999). The reasons for this are not apparent, since cereal fibre is relatively lipid neutral. Alternative explanations presented to explain diabetes protection may apply here also. Unfortunately, viscous soluble fibres are not eaten in sufficient quantities to allow an assessment of these types of fibre to be made in Western population studies.

Low-glycaemic index diets have also been implicated in reducing the risk of cardiovascular disease. Both the Nurses Health and the Health Professionals studies showed a reduction in the risk of cardiovascular risk when low-glycaemic index diets or low-glycaemic load diets were consumed (Lui *et al.* 1998). These benefits are therefore in addition and probably even stronger than the effects seen with low-glycaemic index diets in reducing diabetes incidence.

Clinically, viscous rather than insoluble fibres have long been known to reduce serum lipids in both normal and hyperlipidaemic subjects (Jenkins *et al.* 1975; Braaten *et al.* 1994). Viscous fibres may therefore play a key role in dietary cholesterol reduction and be part of the dietary portfolio of food and lifestyle factors, which if additive could reduce serum lipids to the same extent as currently used drug therapies (Table 13.1) (Jenkins and Kendall 1999). Diets containing low-glycaemic index foods have also been associated with reduced lipids in

Table 13.1 A portfolio of dietary factors for cholesterol reduction.

Dietary component	Dietary changes	Approximate LDL reduction (%)
Saturated fat*	<7% of calories	10
Dietary cholesterol†	<200 mg/day	5
Body weight	Lose 5 kg (10 lb.)	5
Viscous fibre	5–10 g/day	5
Soy protein	25 g/day	5
Plant sterols‡	1–3 g/day	5
Total	Full portfolio‡	35

* Reduce *trans* fatty acid as close to zero as possible.

† Depending on the sterol or stanol.

‡ Assuming that the effects are additive.

LDL, low-density lipoprotein.

hyperlipidaemic subjects. Reductions occurred especially in serum triglycerides, and there was either no change, or a tendency to higher HDL cholesterol levels (Jenkins *et al.* 1987). It is therefore relevant that assessment of British adults and the NHANES III data have shown that the lower the diet glycaemic index, the higher the HDL cholesterol (Ford and Liu 1999; Frost *et al.* 1999), suggesting a further reason for coronary heart disease (CHD) risk reduction on a low-glycaemic index diet. It may be that the strongest case for fibre in CHD reduction lies in the ability of fibre to reduce the glycaemic index of foods.

Cancer

It has been suggested that insulin and insulin-like growth factors may be important in the promotion of cancers (McKeown-Eyssen 1994; Giovannucci 1995).

In this respect, it is worth noting that in a large Italian case-control study, glycaemic index was negatively related to colon cancer (Augustin *et al.* 2000). This opens the door to assessment of the possible role of glycaemic index in other malignancies. Again, the association of higher fibre with low glycaemic index may be the reason for the fibre effect. It may also be failure to assess this association which has led to negative findings in the past, and the lack of glycaemic effect may be the reason for the poor showing of wheat bran in recent trials in relation to polyp recurrence prevention (Alberts *et al.* 2000; Schatzkin *et al.* 2000).

13.4 Conclusion

An important feature by which fibre may act is by reducing the rate of absorption. One effect which fibre and fibre-like substances may have on the diet is to convert the carbohydrate component of foods into a slow release form that requires less insulin for tissue uptake, and also increasing the elimination of bile acids in the faeces. These factors, in addition to altered colonic short-chain fatty acid profiles, may be key to the benefits of high-fibre diets on carbohydrate metabolism, with potential benefits in diabetes, cardiovascular disease and cancer reduction.

References

- Alberts, D.S., Martinez, M.E., Roe, D.J., *et al.* (2000) Lack of effect of a high-fiber cereal supplement on the recurrence of colorectal adenomas. *New England Journal of Medicine*, **342**, 1156–1162.
- Anderson, J.W., Story, L., Sieling, B., Chen, W.J.L., Petro, M.S. & Story, J. (1984) Hypocholesterolemic effects of oat bran or bean intake for hypercholesterolemic men. *American Journal of Clinical Nutrition*, **40**, 1146–1155.
- Augustin, L.S., Jenkins, D.J., Parpinel, M. & Franceschi, S. (2000) Dietary glycaemic load and colorectal cancer risk. *FASEB Journal*, **14**, A771 (abstract).
- Axelsen, M. (1999) *Nocturnal and postprandial metabolism in diabetes*. PhD thesis, Goteborg University.
- Bertelsen, J., Christiansen, C., Thomson, C., *et al.* (1993) Effect of meal frequency on blood glucose insulin and free fatty acids in NIDDM subjects. *Diabetes Care*, **16**, 3–7.
- Blackburn, N.A., Redfern, J.S., Jarjis, H., *et al.* (1984) The mechanism of action of guar gum in improving glucose tolerance in man. *Clinical Science*, **66**, 329–336.
- Bosello, O., Struzzi, R., Armellini, F., Micciolo, R. & Seuro, L.A. (1980) Glucose tolerance and blood lipids in brain fed patients with impaired glucose tolerance. *Diabetes Care*, **3**, 46–49.
- Braaten, J.T., Wood, P.J., Scott, F.W., *et al.* (1994) Oat beta glucan reduces blood cholesterol concentration in hypercholesterolemic subjects. *European Journal of Clinical Nutrition*, **48**, 465–474.
- Burkitt, D.P. & Spiller, G. (1993) Dietary fibre: from early hunter-gatherers to the 1990s. *CRC Handbook of Dietary Fiber in Human Nutrition*, 2nd edn. (ed. G. Spiller), pp. 3–6. CRC Press, Boca Raton, FL.
- Englyst, K.N., Englyst, H.N., Husdon, G.J., Cole, T.J. & Cummings, J.H. (1999) Rapidly available glucose in foods: an *in vitro* measurement which reflects the glycemic response. *American Journal of Clinical Nutrition*, **69**, 448–454.
- Flourie, B., Vidor, N., Florent, C.H. & Berrier, J.J. (1984) Effect of pectin on jejunal glucose absorption and unstirred layer thickness in normal man. *Gut*, **25**, 936–941.
- Ford, E. & Liu, S. (1999) Glycemic index, glycemic load, and serum high density lipoprotein (HDL) concentration among United States Adults. *Circulation*, **100**, 4594 (abstract).
- Frost, G., Leeds, A.R., Dore, C., Madeiros, S., Brading, S. & Dornhorst, A. (1999) Glycaemic index as a determinant of serum HDL-cholesterol concentration. *Lancet*, **353**, 1045–1048.
- Gerriello, A., Lizzio, S., Borlotti, N., Russo, A., Metz, E., Tonutti, L., Crescentini, A. & Tabogon, C. (1998) Meal-generated oxidative stress in type 2 diabetes patients. *Diabetes Care*, **21**, 1529–1533.
- Giovannucci, E. (1995) Insulin and colon cancer. *Cancer Causes and Control*, **6**, 164–179.
- Jacobs, D.R., Meyer, K.A., Kushi, L.H. & Folsom, A.R. (1998) Whole grain intake may reduce the risk of ischemic heart disease death in post menopausal women: the Iowa Women's Health Study. *American Journal of Clinical Nutrition*, **68**, 248–257.
- Jarvi, A.E., Bjorek, I.E., Karlstrom, B.E., Asp, N-G.L., Grandfeldt, Y.E. & Vessby, B.O.H. (1999) Improved glycemic control and lipid profile and normalised fibrinolytic activity on a low glycemic index diet in type 2 diabetic patients. *Diabetes Care*, **22**, 10–18.
- Jenkins, D.J.A. & Kendall, C.W. (1999) Plant sterols, health claims and strategies to reduce cardiovascular disease risk. *Journal of the American College of Nutrition*, **18**, 559–562.
- Jenkins, D.J.A., Leeds, A.R., Newton, C. & Cummings, J.H. (1975) Effect of pectin, guar gum and wheat fibre on serum-cholesterol. *Lancet*, **1**, 1155.
- Jenkins, D.J.A., Wolever, T.M.S., Leeds, A.R., *et al.* (1978) Dietary fibres, fibre analogues and glucose tolerance: importance of viscosity. *British Medical Journal*, **1**, 1372–1374.
- Jenkins, D.J.A., Bloom, S.R., Albuquerque, R.H., *et al.* (1980) Pectin and complications after post-gastric surgery: normalization of post-prandial glucose and endocrine responses. *Gut*, **21**, 574–579.
- Jenkins, D.J.A., Ghafari, H., Wolever, T.M.S., *et al.* (1982a) Relationship between rate of digestion of foods and postprandial glycemia. *Diabetologia*, **22**, 450–455.
- Jenkins, D.J.A., Wolever, T.M.S., Taylor, R., *et al.* (1982b) Slow release carbohydrate improves second meal tolerance. *American Journal of Clinical Nutrition*, **35**, 1339–1346.
- Jenkins, D.J.A., Wolever, T.M.S., Kalmusky, J., *et al.* (1987) Low glycemic index diets in hyperlipidemia: use of traditional starchy foods. *American Journal of Clinical Nutrition*, **46**, 66–71.
- Jenkins, D.J.A., Wolever, T.M.S., Ocana, A.M., *et al.* (1990) Metabolic effects of reducing rate of glucose ingestion by single bolus versus continuous sipping. *Diabetes*, **39**, 775–781.
- Jenkins, D.J.A., Ocana, A., Jenkins, A.L., *et al.* (1992) Metabolic advantages of spreading the nutrient load: effects of meal frequency in non-insulin-dependent diabetes. *American Journal of Clinical Nutrition*, **55**, 461–467.

- Jenkins, D.J.A., Wolever, T.M.S., Vidgen, E., *et al.* (1997) Effect of psyllium in hypercholesterolemia at two monounsaturated fatty acid intakes. *American Journal of Clinical Nutrition*, **66**, 1524-1533.
- Leeds, A.R., Ralphs, D.N.L., Ebied, F., Metz, G. & Dilawari, J.P. (1981) Pectin in the dumping syndrome: reduction of symptoms and plasma volume changes. *Lancet*, **1**, 1075.
- Lia, A., Hallman, G., Sandberg, A.-S., Sandberg, B., Aman, P. & Anderson, H. (1995) Oat beta-glucan increases bile acid excretion and a barley rich fraction increases cholesterol excretion in ileostomy subjects. *American Journal of Clinical Nutrition*, **62**, 1245-1251.
- Ludwig, D., Majzoub, J., Al-Zahrani, A., Dallal, G., Blanco, I. & Roberts, S. (1999a) High glycemic index foods, overeating and obesity. *Pediatrics*, **103**, E26, 656.
- Ludwig, D., Pereira, M.A., Kroenke, J., *et al.* (1999b) Dietary fibre, weight gain and cardiovascular disease risk factors in young adults. *Journal of the American Medical Association*, **282**, 1539-1546.
- Lui, S., Willett, W., Stampfer, M., *et al.* (1998) A prospective study of dietary glycemic load, carbohydrate and risk of coronary heart disease in US women. *FASEB Journal*, **12**, A260 (abstract).
- Lui, S., Stampfer, M., Hu F., *et al.* (1999) Whole grain consumption and risk of coronary heart disease: results from the nurses health study. *American Journal of Clinical Nutrition*, **70**, 412-419.
- McKeown-Eyssen, G. (1994) Epidemiology of colorectal cancer revisited, are serum triglycerides and/or plasma glucose associated with risk? *Cancer Epidemiology Biomarkers Prevention*, **3**, 687-695.
- Metz, P.J., Braaton, J.T., Scott, F.W., Riedel, K.D., Wolynetz, S. & Collins, M.W. (1994) Effect of dose and modification of viscous properties of oat gum on plasma glucose and insulin following an oral glucose load. *British Journal of Nutrition*, **72**, 731-743.
- Salmeron, J., Ascherio, A., Remm, E.B., *et al.* (1997a) Dietary fiber, glycemic load and risk of NIDDM in men. *Diabetes Care*, **20**, 545-550.
- Salmeron, J., Manson, J.E., Stampfer, M.J., Colditz, G.A., Wing, A.C. & Willett, W.C., (1997b) Dietary fibre, glycemic load and risk of non-insulin dependent diabetes in women. *Journal of the American Medical Association*, **277**, 472-477.
- Schatzkin, A., Lanza, E., Corle, D., *et al.* (2000) Lack of effect of a low fat, high fiber diet on recurrence of colorectal adenomas. Polyp Prevention Trial Study Group. *New England Journal of Medicine*, **342**, 1149-1155.
- Trowell, H. (1993) Development of the dietary fibre hypothesis of diabetes mellitus. *CRC Handbook of Dietary Fibre in Human Nutrition*, 2nd edn. (ed. G. Spiller), pp. 439-441. CRC Press, Boca Raton, FL.
- Wolever, T.M.S., Jenkins, D.J.A., Ocana, A., Rao, A.V. & Collier, G. (1988) Second-meal effect: low glycemic-index foods eaten at dinner improve subsequent breakfast glycemic response. *American Journal of Clinical Nutrition*, **48**, 1041-1047.